$%^Dialog;HighlightOn=*;HighlightOff=*;

Connecting via Winsock to Dialog

Logging in to Dialog

Trying 31060000009998...Open

DIALOG INFORMATION SERVICES
PLEASE LOGON:
****
ENTER PASSWORD:
****
Welcome to DIALOG

Dialog level 04.11.00D

Last logoff: 20may04 12:57:14
Logon file405 01jul04 11:43:31
*** ANNOUNCEMENT ***
***

--File 654 - US published applications from March 15, 2001 to the
present are now online. Please see HELP NEWS 654 for details.

***
--File 581 - The 2003 annual reload of Population Demographics is
complete. Please see Help News581 for details.

***
--File 990 - NewsRoom now contains February 2004 to current records.
File 992 - NewsRoom 2003 archive has been newly created and contains
records from January 2003. The oldest months's records roll out of
File 990 and into File 992 on the first weekend of each month.
To search all 2003 records BEGIN 990, 992, or B NEWS2003, a new
OneSearch category.
***
--Connect Time joins DialUnits as pricing options on Dialog.
See HELP CONNECT for information.
***
***
--SourceOne patents are now delivered to your email inbox as PDF replacing TIFF delivery. See HELP SOURCE1 for more
information.
***
--Important Notice to Freelance Authors--
See HELP FREELANCE for more information
***

NEW FILES RELEASED
***MetalBase (File 36)
***AeroBase (File 104)
***DIOGENES: Adverse Drug Events Database (File 181)
***World News Connection (File 985)
***Dialog NewsRoom - 2003 Archive (File 992)
***TRADEMARKSCAN-Czech Republic (File 680)
***TRADEMARKSCAN-Hungary (File 681)
***TRADEMARKSCAN-Poland (File 682)
***
UPDATING RESUMED
***
RELOADED
***Toxfile (File 156)
***Medline (Files 154-155)
***Population Demographics -(File 581)
***CLAIMS Citation (Files 220-222)

REMOVED
***

>>> Enter BEGIN HOMEBASE for Dialog Announcements <<<
>>> of new databases, price changes, etc. <<<
****
HILIGHT set on as '*'
KWIC is set to 50.
* * * *
SYSTEM:HOME
Cost is in DialUnits
Menu System II: D2 version 1.7.9 term=ASCII
*** DIALOG HOMEBASE(SM) Main Menu ***

Information:
1. Announcements (new files, reloads, etc.)
2. Database, Rates, & Command Descriptions
3. Help in Choosing Databases for Your Topic
4. Customer Services (telephone assistance, training, seminars, etc.)
5. Product Descriptions

Connections:
6. DIALOG(R) Document Delivery
7. Data Star(R)

/H = Help /L = Logoff /NOMENU = Command Mode

Enter an option number to view information or to connect to an online
service. Enter a BEGIN command plus a file number to search a database
(e.g., B1 for ERIC).
? b 410

```
     01jul04 11:43:33 User217743 Session D640.1
        $0.00   0.164 DialUnits FileHomeBase
    $0.00  Estimated cost FileHomeBase
    $0.00  Estimated cost this search
    $0.00  Estimated total session cost   0.164 DialUnits

File 410:Chronolog(R) 1981-2004/May
     (c) 2004 The Dialog Corporation

    Set  Items  Description
    ---  -----  -----------
? set hi *;set hi *
HILIGHT set on as '*'*
*HILIGHT set on as '*'
? b 155
     01jul04 11:43:41 User217743 Session D640.2
        $0.00   0.078 DialUnits File410
    $0.00  Estimated cost File410
    $0.03  TELNET
    $0.03  Estimated cost this search
    $0.03  Estimated total session cost   0.242 DialUnits

File 155:MEDLINE(R) 1966-2004/Jun W2
     (c) format only 2004 The Dialog Corp.
*File 155: Medline has been reloaded. Accession numbers
have changed. Please see HELP NEWS 154 for details.

    Set  Items  Description
    ---  -----  -----------
? s infliximab
    S1   1282  INFLIXIMAB
? s etanercept
    S2    559  ETANERCEPT
? s s1 or s2
        1282  S1
         559  S2
    S3   1591  S1 OR S2
? s s3 and (immobilize? or solid()support or affinity)
        1591  S3
       26568  IMMOBILIZE?
       79595  SOLID
     3620501  SUPPORT
        1214  SOLID(W)SUPPORT
      183087  AFFINITY
    S4      9  S3 AND (IMMOBILIZE? OR
SOLID()SUPPORT OR AFFINITY)
? t s4/3,ab/all

 4/3,AB/1
DIALOG(R)File 155:MEDLINE(R)
(c) format only 2004 The Dialog Corp. All rts. reserv.

15841502   PMID: 14632813
 *Infliximab* for hidradenitis suppurativa.
 Sullivan T P; Welsh E; Kerdel F A; Burdick A E; Kirsner R
S
  Department of Dermatology and Cutaneous Surgery,
University of Miami
School of Medicine/Cedars Medical Center, 1400 NW
12th Ave. 6 South
Dermatology, Miami, FL 33136, USA.
  British journal of dermatology (England)  Nov 2003, 149
(5) p1046-9,
ISSN 0007-0963  Journal Code: 0004041
  Document type: Journal Article
  Languages: ENGLISH
  Main Citation Owner: NLM
  Record type: Completed
  BACKGROUND: Hidradenitis suppurativa (HS) is a
chronic disease
characterized by significant morbidity. Current medical
therapies are only
minimally effective at treating the disease. *Infliximab* is
a chimeric
monoclonal antibody with high *affinity* for tumour
necrosis factor
(TNF)-alpha. TNF-alpha is known to induce
proinflammatory cytokines and may
play an important role in the therapy of a number of
disparate inflammatory
disorders. *Infliximab* has shown promise for the therapy
of rheumatoid
arthritis and psoriasis. OBJECTIVES: Retrospectively
to evaluate the
effectiveness of *infliximab* for the treatment of HS.
METHODS: A
retrospective chart review was performed for
patients who received
*infliximab* at the University of Miami Department of
Dermatology.
Patients were contacted and asked retrospectively to
rate their disease
activity immediately prior to and after therapy.
RESULTS: Patients'
self-reported disease activity scores were significantly
decreased (P =
0.0001, paired t-test) following *infliximab* infusion. This
correlated
with physician-observed clinical improvement.
CONCLUSIONS: *Infliximab*
is a promising agent for the treatment of HS. These initial
results suggest
that *infliximab* is associated with objective and
subjective
improvement in HS. Further controlled studies of the
efficacy of
*infliximab* and its effect on the course of the disease
are warranted.

 4/3,AB/2
DIALOG(R)File 155:MEDLINE(R)
(c) format only 2004 The Dialog Corp. All rts. reserv.
```

15008755 PMID: 12171504
Soluble cytokine receptors in biological therapy.
Fernandez-Botran Rafael; Crespo Fabian A; Sun Xichun
Department of Pathology & Laboratory Medicine, School of Medicine,
University of Louisville, Louisville, KY 40292, USA. Rafael@louisville.edu
Expert opinion on biological therapy (England) Aug 2002, 2 (6)
p585-605, ISSN 1471-2598 Journal Code: 101125414
Document type: Journal Article
Languages: ENGLISH
Main Citation Owner: NLM
Record type: In Data Review
Due to their fundamental involvement in the pathogenesis of many
diseases, cytokines constitute key targets for biotherapeutic approaches.
The discovery that soluble forms of cytokine receptors are involved in the
endogenous regulation of cytokine activity has prompted substantial
interest in their potential application as immunotherapeutic agents. As
such, soluble cytokine receptors have many advantages, including
specificity, low immunogenicity and high *affinity* . Potential
disadvantages, such as low avidity and short in vivo half-lifes, have been
addressed by the use of genetically-designed receptors, hybrid proteins or
chemical modifications. The ability of many soluble cytokine receptors to
inhibit the binding and biological activity of their ligands makes them
very specific cytokine antagonists. Several pharmaceutical companies have
generated a number of therapeutic agents based on soluble cytokine
receptors and many of them are undergoing clinical trials. The most
advanced in terms of clinical development is *etanercept* (Enbrel,
Immunex), a fusion protein between soluble TNF receptor Type II and the Fc
region of human IgG1. This TNF-alpha; antagonist was the first soluble
cytokine receptor to receive approval for use in humans. In general, most
agents based on soluble cytokine receptors have been safe, well-tolerated
and have shown only minor side effects in the majority of patients. Soluble
cytokine receptors constitute a new generation of therapeutic agents with
tremendous potential for applications in a wide variety of human diseases.
Two current areas of research are the identification of their most
promising applications and characterisation of their long-term effects.

4/3,AB/3
DIALOG(R)File 155:MEDLINE(R)

15008454 PMID: 12110154
How does *infliximab* work in rheumatoid arthritis?
Maini Ravinder N; Feldmann Marc
The Kennedy Institute of Rheumatology Division, Imperial College of
Science Technology and Medicine, London, UK. r.maini@ic.ac.uk
Arthritis research (England) 2002, 4 Suppl 2 pS22-8, ISSN 1465-9905
Journal Code: 100913255
Document type: Journal Article
Languages: ENGLISH
Main Citation Owner: NLM
Record type: In Data Review
Since the initial characterization of tumor necrosis factor alpha
(TNFalpha), it has become clear that TNFalpha has diverse biologic
activity. The realization that TNFalpha plays a role in rheumatoid
arthritis (RA) has led to the development of anti-TNF agents for the
treatment of RA. *Infliximab* , a chimeric monoclonal antibody that
specifically, and with high *affinity* , binds to TNFalpha and
neutralizes the cytokine, is currently approved for the treatment of RA and
Crohn's disease, another immune-inflammatory disorder. In addition to
establishing the safety and efficacy of *infliximab*, clinical research
has also provided insights into the complex cellular and cytokine-dependent
pathways involved in the pathophysiology of RA, including evidence that
supports TNFalpha involvement in cytokine regulation, cell recruitment,
angiogenesis, and tissue destruction.

4/3,AB/4
DIALOG(R)File 155:MEDLINE(R)

12628340 PMID: 7538333
The mouse/human chimeric monoclonal antibody cA2 neutralizes TNF in vitro

and protects transgenic mice from cachexia and TNF lethality in vivo.
Siegel S A; Shealy D J; Nakada M T; Le J; Woulfe D S; Probert L; Kollias
G; Ghrayeb J; Vilcek J; Daddona P E
Department of Immunology, Centocor, Inc., Malvern PA 19355, USA.
Cytokine (UNITED STATES) Jan 1995, 7 (1) p15-25, ISSN 1043-4666
Journal Code: 9005353
Document type: Journal Article
Languages: ENGLISH
Main Citation Owner: NLM
Record type: Completed
The pleiotropic cytokine tumour necrosis factor-alpha (TNF) is thought to
play a central role in infectious, inflammatory and autoimmune diseases.
Critical to the understanding and management of TNF-associated pathology is
the development of highly specific agents capable of modifying TNF
activity. We evaluated the ability of a high *affinity* mouse/human
chimeric anti-TNF monoclonal antibody (cA2) to neutralize the in vitro and
in vivo biological effects of TNF. cA2 inhibited TNF-induced mitogenesis
and IL-6 secretion by human fibroblasts, TNF-priming of human neutrophils,
and the stimulation of human umbilical vein endothelial cells by TNF as
measured by the expression of E-selectin, ICAM-1 and procoagulant activity.
cA2 also specifically blocked TNF-induced adherence of human neutrophils to
an endothelial cell monolayer. Receptor binding studies suggested that
neutralization resulted from cA2 blocking of TNF binding to both p55 and
p75 TNF receptors on the cells. In vivo, repeated administration of cA2 to
transgenic mice that constitutively express human TNF reversed the
cachectic phenotype and prevented subsequent mortality. These results
demonstrated that cA2 effectively neutralized a broad range of TNF
biological activities both in vitro and in vivo.

4/3,AB/5
DIALOG(R)File 155:MEDLINE(R)

12055469 PMID: 12379627
Infliximab treatment for rheumatic disease: clinical and
radiological efficacy.
St Clair E W
Department of Medicine, Division of Rheumatology, Duke University Medical
Center, Durham, NC, USA. stcla003@mc.duke.edu
Annals of the rheumatic diseases (England) Nov 2002, 61 Suppl 2
pii67-9, ISSN 0003-4967 Journal Code: 0372355
Document type: Journal Article; Review; Review, Tutorial
Languages: ENGLISH
Main Citation Owner: NLM
Record type: Completed
Infliximab is a chimeric anti-tumour necrosis factor alpha
(TNFalpha) monoclonal antibody with high *affinity* and binding
specificity for human TNFalpha. Results from several well designed,
controlled clinical trials show repeated infusions of *infliximab* with
concomitant methotrexate (MTX) treatment can reduce the signs and symptoms
of rheumatoid arthritis (RA). This combination of *infliximab* and MTX
also slows the radiological progression of joint damage, decreases
functional disability, and improves qualify of life. These remarkably
positive results have led to the investigation of *infliximab*
treatment for other rheumatic diseases. Recently, controlled studies have
shown treatment with *infliximab* can benefit patients with active
spondyloarthropathy. TNFalpha has fast become an important therapeutic
target in rheumatology.

4/3,AB/6
DIALOG(R)File 155:MEDLINE(R)

11423402 PMID: 11520249
New therapeutic approaches to the management of rheumatoid arthritis.
Hughes L B; Moreland L W
Department of Medicine, Division of Clinical Immunology and Rheumatology,
University of Alabama at Birmingham, 1813 6th Ave South, Birmingham, AL
35294, USA. Laura.Hughes@ccc.uab.edu
BioDrugs - clinical immunotherapeutics, biopharmaceuticals and gene
therapy (New Zealand) 2001, 15 (6) p379-93, ISSN 1173-8804
Journal Code: 9705305
Document type: Journal Article

Languages: ENGLISH
Main Citation Owner: NLM
Record type: Completed
Rheumatoid arthritis (RA) is a common disease that affects up to 1% of the population, and causes significant morbidity and early mortality. The aetiology of RA is unknown; however, in the last 10 to 15 years significant advances in molecular technology have provided a greater understanding of the pathogenesis of the disease. This has led to the development of new approaches to the treatment of RA. The disease modifying antirheumatic oral agent leflunomide inhibits the proliferation of activated T cells that are important in the inflammation and degradation of synovial tissues. The 2 biological agents approved for the treatment of RA, *infliximab* and *etanercept* , are inhibitors of the pro-inflammatory cytokine, tumour necrosis factor-alpha (TNFalpha). *Infliximab* is a chimeric human/mouse monoclonal antibody which is administered by intravenous infusion and binds with high *affinity* to TNFalpha, thereby neutralising its effects. *Etanercept* is a recombinant, soluble TNF receptor molecule which is administered subcutaneously and binds to TNFalpha in the serum rendering it biologically inactive. The protein A immunoadsorption column is a medical device that in conjunction with plasmapheresis can be used in patients with refractory RA. These agents have provided new and effective therapies for the treatment of patients with RA.

4/3,AB/7
DIALOG(R)File 155:MEDLINE(R)

11367282 PMID: 11458980
Immunomodulatory effects of *etanercept* (TNFR:Fc) and its use in a patient with Crohn's disease.
Srivastava M D
Department of Pediatrics, MetroHealth Medical Center, Cleveland, Ohio 44109, USA.
Research communications in molecular pathology and pharmacology (United States) Jul 2001, 109 (1-2) p125-41, ISSN 1078-0297 Journal Code: 9437512
Document type: Case Reports; Clinical Trial; Journal Article
Languages: ENGLISH
Main Citation Owner: NLM
Record type: Completed
Designer drug *etanercept* (TNFR:Fc) is an inhibitor of TNF-alpha that binds with greater *affinity* than membrane receptors. Its full immunomodulatory effects are unknown. Approved for rheumatoid arthritis, its therapeutic potential in Crohn's disease has yet to be explored. We describe the course of a steroid-dependent patient with Crohn's disease given *etanercept* , and its effects on cytokine protein and mRNA expression and transcription factor activity in human leukocytes. *Etanercept* 25 mg s.c., was given twice weekly for 1 month. Weekly ESR, disease activity index, prednisone requirement, and serum cytokines were determined. In vitro, effects of physiologic concentrations of *etanercept* on cytokine protein and mRNA, and NFKB and GR transcription factor activity, were determined using MOT and U937 cell lines and peripheral blood mononuclear cells. Rapid clinical, biochemical, and immunologic improvement occurred, but obstruction due to stricture developed after 4 weeks. In vitro, constitutive and stimulated production of TNF-beta, IL-1beta, MIP-1beta, and IL-8 by normal mononuclear cells declined with *etanercept* , detectable TNF-alpha increased. MOT TNF-alpha expression tripled, mRNA for IL-12 p40 doubled, GR activity declined in U937 cells, NFKB was unaffected. *Etanercept* has complex immunomodulatory effects, and may be useful in Crohn's disease, but acutely decreased inflammation could worsen stricture.

4/3,AB/8
DIALOG(R)File 155:MEDLINE(R)

11209364 PMID: 11249494
Tumour necrosis factor-alpha blockade: a new era for effective management

of rheumatoid arthritis.
Hamilton K; Clair E W
Division of Rheumatology, Allergy and Clinical Immunology, Department of
Medicine, Duke University Medical Centre, Durham, NC 27710, USA.
Expert opinion on pharmacotherapy (England) Jul 2000, 1 (5) p1041-52
, ISSN 1465-6566 Journal Code: 100897346
Document type: Journal Article; Review; Review, Tutorial
Languages: ENGLISH
Main Citation Owner: NLM
Record type: Completed
Tumour necrosis factor (TNF)-alpha inhibitors have emerged as a new
treatment option for rheumatoid arthritis (RA). The scientific rationale
for targeting TNF-alpha in RA derives from extensive work in the
laboratory, showing the importance of this pro-inflammatory cytokine as a
mediator of joint inflammation. Proof of principle has now been firmly
established in clinical trials where TNF-alpha inhibitors have been shown
to decrease the signs and symptoms of joint inflammation and slow
radiological progression of joint damage. Presently, the two TNF-alpha
inhibitors available for use in RA are *etanercept* and
infliximab. *Etanercept* is a soluble TNF receptor: Fc fusion
protein that competes with the endogenous TNF receptors for TNF-alpha
binding. *Infliximab* is a chimeric anti-TNF-alpha monoclonal antibody,
which also binds with high *affinity* to soluble TNF-alpha.
Etanercept and *infliximab* will be rapidly incorporated into
current treatment paradigms, which call for early and intensive treatment
of RA using disease-modifying antirheumatic drugs (DMARDs), such as
methotrexate, sulfasalazine and hydroxychloroquine. A major drawback to the
widespread use of these biologics is their high costs. Some patients with
limited financial means may be denied access to these effective
anti-inflammatory agents. Moreover, long-term experience with TNF-alpha
inhibitor therapy has been limited and concerns linger about the
possibility that *etanercept* and *infliximab* may cause unforeseen
side effects or increase the risk for opportunistic infection. Despite
these caveats, TNF-alpha inhibitors represent a major advance for the
treatment of RA and will likely spawn new indications for anti-TNF-alpha
therapy and the development of novel therapeutic compounds with similar
biological activity.

4/3,AB/9
DIALOG(R)File 155:MEDLINE(R)

10921458 PMID: 11060691
Soluble cytokine receptors: novel immunotherapeutic agents.
Fernandez-Botran R
Departments of Pathology & Laboratory Medicine and Microbiology &
Immunology, School of Medicine, University of Louisville, Louisville, KY
40292, USA.
Expert opinion on investigational drugs (ENGLAND) Mar 2000, 9 (3)
p497-514, ISSN 1354-3784 Journal Code: 9434197
Document type: Journal Article; Review; Review, Academic
Languages: ENGLISH
Main Citation Owner: NLM
Record type: Completed
Being mediators of immune and inflammatory reactions, abnormal or
excessive production of cytokines is often the main cause of the pathology
in many types of disease. Targeting cytokines by means of inhibitory drugs
may thus offer a valid therapeutic approach in particular diseases. Soluble
forms of cytokine receptors (sCR) normally participate in the control of
cytokine activity in vivo by inhibiting the ability of cytokines to bind
their membrane receptors and from generating a biological response. The
ability of sCR to act as cytokine inhibitors, coupled to their specificity,
high affinities and low immunogenicities have prompted considerable
interest in their use as immunotherapeutic agents. In fact, many types of
sCR have been shown to inhibit the biological activity of their cytokines
in vitro and in different experimental models. Several sCR, particularly
the soluble TNF receptors sTNFR-I (p55) and sTNFR-II (p75), have been

modified by linking them to the Fc portion of human immunoglobulin (e.g., 'immunoadhesins') or by the addition of polyethylene-glycol (PEG) (e.g., 'PEGylation'), in order to enhance their *affinity* and/or biological half-life. These agents have shown significant therapeutic value in clinical trials of patients with rheumatoid arthritis (RA). Indeed, a sTNFR-II:Fc hybrid molecule (*etanercept*), the first sCR-derived therapeutic agent to receive approval for human use, is already utilised for the treatment of some forms of RA. Additional applications of this drug in other inflammatory conditions are currently being evaluated, while another sCR-derived agent, a human sIL-4R, is undergoing trials for the treatment of asthma. Many other sCR, such as sIL-1R, sIL-5R, sIFNgammaR, may also have significant potential for the treatment of a wide variety of human diseases.

? ds

```
Set    Items  Description
S1     1282   INFLIXIMAB
S2      559   ETANERCEPT
S3     1591   S1 OR S2
S4        9   S3 AND (IMMOBILIZE? OR
SOLID()SUPPORT OR AFFINITY)
? s s3 and serum
         1591 S3
       514330 SERUM
    S5    81 S3 AND SERUM
? s s5 and ex()vivo
          81 S5
       22634 EX
      348124 VIVO
       15309 EX(W)VIVO
    S6     0 S5 AND EX()VIVO
? s s5 and dialyze
          81 S5
          65 DIALYZE
    S7     0 S5 AND DIALYZE
? s s5 and dialysis
          81 S5
       86686 DIALYSIS
    S8     1 S5 AND DIALYSIS
? t s8/3,ab/

 8/3,AB/1
DIALOG(R)File 155:MEDLINE(R)
(c) format only 2004 The Dialog Corp. All rts. reserv.

11345614   PMID: 11434729
```

Treatment of complicated sarcoidosis with *infliximab* anti-tumor necrosis factor-alpha therapy.

Yee A M; Pochapin M B
Hospital for Special Surgery and New York Presbyterian Hospital, Weill Medical College of Cornell University, New York, New York, USA.

Annals of internal medicine (United States) Jul 3 2001, 135 (1) p27-31, ISSN 0003-4819 Journal Code: 0372351
Comment in Ann Intern Med. 2002 Aug 20;137(4) 296-7; author reply 296-7; Comment in PMID 12186527; Comment in Ann Intern Med. 2002 Aug 20;137(4):296-7; author reply 296-7; Comment in PMID 12186528
Document type: Case Reports; Journal Article
Languages: ENGLISH
Main Citation Owner: NLM
Record type: Completed

BACKGROUND: Tumor necrosis factor-alpha (TNF-alpha) may have an important role in the clinical exacerbation of sarcoidosis. OBJECTIVE: To treat sarcoidosis with *infliximab* , a chimeric human-murine anti-human TNF-alpha monoclonal antibody. DESIGN: Case report. SETTING: U.S. academic medical center. PATIENT: A 72-year-old woman with sarcoidosis presenting with severe protein-losing enteropathy, hypoalbuminemia, and proximal myopathy who had not responded adequately to corticosteroid therapy and whose clinical course was further complicated by acute tubular necrosis and renal failure requiring long-term hemodialysis. INTERVENTION: Intravenous infusion of *infliximab* , 5 mg/kg of ideal body weight; infusion was repeated at 2 and 6 weeks. MEASUREMENTS: Clinical response of enteropathic and myopathic symptoms and *serum* albumin level. RESULTS: Enteropathic and myopathic symptoms resolved after *infliximab* therapy, and the *serum* albumin level also improved. However, the clinical course was complicated by the development of a hypercoagulable state associated with circulating anticardiolipin antibodies, which prompted discontinuation of *infliximab* therapy. CONCLUSIONS: *Infliximab* therapy was successful in a patient with sarcoidosis. Tumor necrosis factor-alpha may

be an important mediator of clinical disease in sarcoidosis and could be an
attractive target for therapeutic intervention. However, *infliximab*
may cause adverse effects associated with cytokine cascade manipulation.
? t s8/kwic/

8/KWIC/1
DIALOG(R)File 155:(c) format only 2004 The Dialog Corp. All rts. reserv.

Treatment of complicated sarcoidosis with *infliximab* anti-tumor
necrosis factor-alpha therapy.
BACKGROUND: Tumor necrosis factor-alpha (TNF-alpha) may have an important
role in the clinical exacerbation of sarcoidosis. OBJECTIVE: To treat
sarcoidosis with *infliximab* , a chimeric human-murine anti-human
TNF-alpha monoclonal antibody. DESIGN: Case report. SETTING: U.S. academic
medical center. PATIENT: A 72-year-old woman...

... to corticosteroid therapy and whose clinical course was further
complicated by acute tubular necrosis and renal failure requiring long-term
hemodialysis. INTERVENTION: Intravenous infusion of *infliximab*, 5
mg/kg of ideal body weight; infusion was repeated at 2 and 6 weeks.
MEASUREMENTS: Clinical response of enteropathic and myopathic symptoms and
serum albumin level. RESULTS: Enteropathic and myopathic symptoms
resolved after *infliximab* therapy, and the *serum* albumin level
also improved. However, the clinical course was complicated by the
development of a hypercoagulable state associated with circulating
anticardiolipin antibodies, which prompted discontinuation of
infliximab therapy. CONCLUSIONS: *Infliximab* therapy was
successful in a patient with sarcoidosis. Tumor necrosis factor-alpha may
be an important mediator of clinical disease in sarcoidosis and could be an
attractive target for therapeutic intervention. However, *infliximab*
may cause adverse effects associated with cytokine cascade manipulation.
...; Lung Diseases--complications--CO; Muscle Weakness--complications--CO
; Muscle Weakness--drug therapy--DT; Protein-Losing Enteropathies
--complications--CO; Protein-Losing Enteropathies--drug therapy--DT; Renal
Dialysis; Sarcoidosis--blood--BL; Sarcoidosis--complications--CO;
Serum Albumin--metabolism--ME; Thrombosis--chemically induced--CI
Chemical Name: Antibodies, Monoclonal; *Serum* Albumin; Tumor
Necrosis Factor; *infliximab*
? s s5 and (ultrafiltration or ultrapheresis)
81 S5
10931 ULTRAFILTRATION
4 ULTRAPHERESIS
S9 0 S5 AND (ULTRAFILTRATION OR ULTRAPHERESIS)
? s s5 and apheresis
81 S5
2691 APHERESIS
S10 0 S5 AND APHERESIS
? b 411
01jul04 11:53:08 User217743 Session D640.3
$4.54 1.417 DialUnits File155
$0.05 1 Type(s) in Format 95 (KWIC)
$2.10 10 Type(s) in Format 4 (UDF)
$2.15 11 Types
$6.69 Estimated cost File155
$2.49 TELNET
$9.18 Estimated cost this search
$9.21 Estimated total session cost 1.659 DialUnits
File 411:DIALINDEX(R)

DIALINDEX(R)
(c) 2004 The Dialog Corporation plc

*** DIALINDEX search results display in an abbreviated ***
*** format unless you enter the SET DETAIL ON command. ***
? set files biochem
>>> 162 is unauthorized
>>>1 of the specified files is not available
You have 21 files in your file list.
(To see banners, use SHOW FILES command)
? s (etanercept or infliximab) and apheresis

Your SELECT statement is:
s (etanercept or infliximab) and apheresis

Items	File
2	34: SciSearch(R) Cited Ref Sci_1990-2004/Jun W4
12	73: EMBASE_1974-2004/Jun W4
2	155: MEDLINE(R)_1966-2004/Jun W2

3 files have one or more items; file list includes 21 files.

? rf
Your last SELECT statement was:
S (ETANERCEPT OR INFLIXIMAB) AND APHERESIS

Ref	Items	File
N1	12	73: EMBASE_1974-2004/Jun W4
N2	2	34: SciSearch(R) Cited Ref Sci_1990-2004/Jun W4
N3	2	155: MEDLINE(R)_1966-2004/Jun W2
N4	0	5: Biosis Previews(R)_1969-2004/Jun W4
N5	0	6: NTIS_1964-2004/Jun W4
N6	0	40: Enviroline(R)_1975-2004/May
N7	0	50: CAB Abstracts_1972-2004/May
N8	0	65: Inside Conferences_1993-2004/Jun W4
N9	0	71: ELSEVIER BIOBASE_1994-2004/Jun W3
N10	0	94: JICST-EPlus_1985-2004/Jun W1

3 files have one or more items; file list includes 21 files.

- Enter P or PAGE for more -
? b n3, n1
01jul04 11:54:09 User217743 Session D640.4
$0.57 0.254 DialUnits File411
$0.57 Estimated cost File411
$0.50 TELNET
$1.07 Estimated cost this search
$10.28 Estimated total session cost 1.914 DialUnits

SYSTEM:OS - DIALOG OneSearch
File 155:MEDLINE(R) 1966-2004/Jun W2
(c) format only 2004 The Dialog Corp.
*File 155: Medline has been reloaded. Accession numbers have changed. Please see HELP NEWS 154 for details.
File 73:EMBASE 1974-2004/Jun W4
(c) 2004 Elsevier Science B.V.

Set	Items	Description
? s (etanercept or infliximab) and apheresis		
	2611	ETANERCEPT
	4228	INFLIXIMAB
	5977	APHERESIS
S1	14	(ETANERCEPT OR INFLIXIMAB) AND APHERESIS
? rd		
...completed examining records		
S2	13	RD (unique items)

? t s2/3,ab/all

2/3,AB/1 (Item 1 from file: 155)
DIALOG(R)File 155:MEDLINE(R)
(c) format only 2004 The Dialog Corp. All rts. reserv.

16254114 PMID: 14727131
Medical approaches and future options in chronic active ulcerative
colitis.
Siveke J T; Folwaczny C
II. Medizinische Klinik, Klinikum rechts der Isar, Technische Universitat
Munchen, Munich, Germany.
International journal of colorectal disease (Germany) Jul 2004, 19
(4) p297-307, ISSN 0179-1958 Journal Code: 8607899
Document type: Journal Article
Languages: ENGLISH
Main Citation Owner: NLM
Record type: In Data Review
BACKGROUND. Immunosuppressive therapy employing purine analogues is the
therapeutic mainstay in patients with chronic active ulcerative colitis.
However, despite therapeutic optimization according to
thiopurine-methyltransferase activity or red blood cell 6-thioguanine
levels, a substantial proportion of patients does not tolerate azathioprine
or 6-mercaptopurine or relapses during this treatment. In the latter
multiple therapeutic regimens comprising 6-thioguanine, cyclosporin or
tacrolimus, methotrexate, cyclophosphamide, *infliximab*, interferons,
heparin, leukocyte *apheresis* , and various other regimens might be
considered aiming at long-term remission. Many of these treatment forms
have only been evaluated in small mostly uncontrolled trials. OBJECTIVE. In
this review existing treatment modalities and future options for patients
with chronic active ulcerative colitis will be discussed focusing on
immunomodulating approaches.

2/3,AB/2 (Item 2 from file: 155)
DIALOG(R)File 155:MEDLINE(R)
(c) format only 2004 The Dialog Corp. All rts. reserv.

11275455 PMID: 11354293
Cost-effectiveness of Prosorba column therapy for rheumatoid arthritis: a
framework for analysis.
Griffiths R I; Slurzberg J E
Project HOPE Center for Health Affairs, Bethesda, Maryland 20814-6133,
USA. rgriffiths@projhope.org
Therapeutic apheresis - official journal of the International Society for

Apheresis and the Japanese Society for Apheresis (United States) Apr 2001
, 5 (2) p105-10, ISSN 1091-6660 Journal Code: 9706703
Document type: Journal Article
Languages: ENGLISH
Main Citation Owner: NLM
Record type: Completed
Apheresis with the Prosorba column is safe and effective for
treating refractory rheumatoid arthritis. It also is resource intensive.
Economic evaluation of Prosorba column therapy could help promote efficient
use of this technology. This article describes a framework and the data
requirements for analyzing the cost-effectiveness of Prosorba column
therapy. Several factors are considered in developing the framework
including the target patient population, treatment alternatives, and
clinical, economic, and quality of life outcomes of alternative treatments.
We propose decision modeling as the appropriate study design because it
provides a flexible framework for combining and analyzing data from
different sources including experimental and nonexperimental studies. The
cost-effectiveness of Prosorba column therapy will depend on the patient
population in which it is used and the other treatment options still
available to these patients. Offsets to the costs of providing Prosorba
column therapy are likely to be largest in treatment-refractory patients
and when this therapy is compared to other expensive new agents such as
etanercept.

2/3,AB/3 (Item 1 from file: 73)
DIALOG(R)File 73:EMBASE

12212177 EMBASE No: 2003319719
Investigational therapies for psoriasis
Cather J.C.; Cather J.C.; Abramovits W.
Dr. J.C. Cather, 5310 Harvest Hill, Dallas, TX 75230 United States
AUTHOR EMAIL: research@texasderm.com
Journal of the American Academy of Dermatology (J. AM. ACAD. DERMATOL.)
(United States) 01 AUG 2003, 49/2 A (S133-S138)
CODEN: JAADD ISSN: 0190-9622
DOCUMENT TYPE: Journal ; Review
LANGUAGE: ENGLISH SUMMARY LANGUAGE: ENGLISH
NUMBER OF REFERENCES: 48

As the pathogenesis of psoriasis is better understood, specific targeted
therapies are being developed. In addition to alefacept, *etanercept*,
efaluzimab, and *infliximab*, discussed in separate articles in this
issue, numerous other investigational therapies are currently in clinical
trials, some of which will likely be approved in the future. We review the
most promising of these therapies and their mechanisms of action.

2/3,AB/4 (Item 2 from file: 73)
DIALOG(R)File 73:EMBASE

12193654 EMBASE No: 2003304874
Islet transplants and impact on secondary diabetic complications: Does
C-peptide protect the kidney?
Shapiro A.M.J.
Dr. A.M.J. Shapiro, Clinical Islet Transplant Program, University of
Alberta, 2000 College Plaza, 8215 112 Street, Edmonton, Alta. T6G 2C8
Canada
AUTHOR EMAIL: amjs@islet.ca
Journal of the American Society of Nephrology (J. AM. SOC. NEPHROL.) (
United States) 01 AUG 2003, 14/8 (2214-2216)
CODEN: JASNE ISSN: 1046-6673
DOCUMENT TYPE: Journal ; Editorial
LANGUAGE: ENGLISH
NUMBER OF REFERENCES: 39

2/3,AB/5 (Item 3 from file: 73)
DIALOG(R)File 73:EMBASE

12010223 EMBASE No: 2003120768
Novel therapies in the treatment of ulcerative colitis
Tulvin J.A.; Kane S.V.
S.V. Kane, Department of Medicine, Division of Gastroenterology,
University of Chicago, 5841 South Maryland Ave., Chicago, IL 60637
United States
AUTHOR EMAIL: skane@medicine.bsd.uchicago.edu
Expert Opinion on Investigational Drugs (EXPERT OPIN. INVEST. DRUGS) (
United Kingdom) 01 MAR 2003, 12/3 (483-490)

CODEN: EOIDE ISSN: 1354-3784
DOCUMENT TYPE: Journal ; Review
LANGUAGE: ENGLISH SUMMARY LANGUAGE: ENGLISH
NUMBER OF REFERENCES: 78

Ulcerative colitis is a chronic inflammatory disease of the colon of unknown cause. Its course is one of relapse and remission and requires therapy for both the induction and maintenance of remission. Progress in the fields of genetics and immunology affords important advances in our understanding of the inflammatory process. Traditional therapy for ulcerative colitis with nonspecific anti-inflammatories remains our gold standard. This review examines the most recent compounds in development for the treatment of ulcerative colitis, including data from early clinical trials and the potential clinical impact of future entities.

2/3,AB/6 (Item 4 from file: 73)
DIALOG(R)File 73:EMBASE

11988299 EMBASE No: 2003098739
Autologous haematopoietic stem cell transplantation in juvenile idiopathic arthritis
Wedderburn L.R.; Abinun M.; Palmer P.; Foster H.E.
Dr. L.R. Wedderburn, Rheumatology Unit, Institute of Child Health, 30 Guilford Street, London WC1N 1EH United Kingdom
AUTHOR EMAIL: l.wedderburn@ich.ucl.ac.uk
Archives of Disease in Childhood (ARCH. DIS. CHILD.) (United Kingdom)
01 MAR 2003, 88/3 (201-205)
CODEN: ADCHA ISSN: 0003-9888
DOCUMENT TYPE: Journal ; Review
LANGUAGE: ENGLISH
NUMBER OF REFERENCES: 46

2/3,AB/7 (Item 5 from file: 73)
DIALOG(R)File 73:EMBASE

11838118 EMBASE No: 2002410807
Treatment of Crohn's disease - The new era
Jewell D.P.
Prof. D.P. Jewell, Gastroenterology Unit, Radcliffe Infirmary, Woodstock Road, Oxford OX2 6HE United Kingdom
Digestive and Liver Disease (DIG. LIVER DIS.) (Italy) 01 OCT 2002,
34/1 (689-691)
CODEN: DLDIF ISSN: 1590-8658
DOCUMENT TYPE: Journal ; Article
LANGUAGE: ENGLISH
NUMBER OF REFERENCES: 12

2/3,AB/8 (Item 6 from file: 73)
DIALOG(R)File 73:EMBASE

11633968 EMBASE No: 2002205544
Successful extracorporeal photochemotherapy for chronic graft-versus-host disease in pediatric patients
Halle P.; Paillard C.; D'Incan M.; Bordigoni P.; Piguet C.; De Lumley L.; Stephan J.L.; Berger M.; Rapatel C.; Demeocq F.; Kanold J.
J. Kanold, U. Bioclin. de Therapie Cellulaire, Serv. d'Hematol./d'Oncol. Pediat., Hotel Dieu, 11, Boulevard Leon Malfreyt, 63003 Clermont-Ferrand France
AUTHOR EMAIL: jkanold@chu-clermontferrand.fr
Journal of Hematotherapy and Stem Cell Research (J. HEMATOTHER. STEM CELL RES.) (United States) 2002, 11/3 (501-512)
CODEN: JHERF ISSN: 1525-8165
DOCUMENT TYPE: Journal ; Article
LANGUAGE: ENGLISH SUMMARY LANGUAGE: ENGLISH
NUMBER OF REFERENCES: 35

A total of 254 extracorporeal photochemotherapy (ECP) procedures were performed in 8 children (median age 10 years; range 5-15) with extensive resistant chronic graft-versus-host disease (GVHD). ECP was carried out in the pediatric environment using a Cobe Spectra separator and UV-MATIC irradiator. A peripheral venous with a single-lumen permanent central catheter access (69% of ECP- *apheresis*) or a dual-lumen permanent central catheter access (26% of ECP-*apheresis*) were used preferentially. A median platelet decrease of 17% (0-71) (p = 0.0001) and median hemoglobin level decrease of 15 g/L (0-31) (p = 0.0001) were noted following each ECP-*apheresis*. However, none of the patients had profound thrombocytopenia or anemia. Two minor episodes of catheter

related-bacteriemia (Staphylococcus aureus) were noted (2310
catheter-days). A negative correlation was found between lymphocyte
collection efficacy (median = 38%) and pre ECP-*apheresis* lymphocyte
count (r = 0.4, p = 0.00001). The median of 5 x 10SUP7 lymphocytes/kg
(0.1-50.10SUP7/kg) was irradiated in each procedure. All patients are alive
and well, and 7/8 experienced a dramatic improvement in their cutaneous
status. Liver and gut disease resolved completely in 4/6 and 5/5 patients,
respectively. In all patients, a concomitant immunosuppressive therapy was
stopped (5/8) or considerably reduced (3/8). Five patients with more than 2
years follow-up after discontinuation of ECP are in remission with no
immunosuppression treatment. They have normal growth rates and normal
school and sport activity. Our study shows that ECP is beneficial, well
tolerated, and can be safely used for chronic GVHD treatment even in young
children with low body weight and a poor performance status. We believe
that having a dedicated pediatric environment together with an experienced,
motivated, and specifically pediatric team is of crucial importance for
improving patient's acceptance of this long-term therapeutic program.

2/3,AB/9 (Item 7 from file: 73)
DIALOG(R)File 73:EMBASE

11617121 EMBASE No: 2002188728
Juvenile dermatomyositis: Recognition and treatment
Reed A.M.; Lopez M.
Dr. A.M. Reed, Division of Rheumatology, Mayo Clinic, 200 First Street
SW, Rochester, MN 55905 United States
AUTHOR EMAIL: reed.ann18@mayo.edu
Paediatric Drugs (PAEDIATR. DRUGS) (New Zealand) 2002, 4/5 (315-321)
CODEN: PTDGF ISSN: 1174-5878
DOCUMENT TYPE: Journal ; Review
LANGUAGE: ENGLISH SUMMARY LANGUAGE: ENGLISH
NUMBER OF REFERENCES: 56

Juvenile dermatomyositis (JDM) is a multisystem disease characterized by acute and chronic lymphocytic inflammation of the skeletal muscle and skin. The disease is marked early in its course by the presence of a vasculopathy or vasculitis, and later by the development of calcinosis. Research has focused on the epidemiology, etiology, and pathogenesis of the disease with, until more recently, limited therapeutic interventions. This article highlights treatment regimens, both traditional and more recent interventions. Traditional treatment for JDM includes high dose corticosteroid treatment with additional agents used in resistant disease or children with unwarranted adverse effects. Traditional therapy begins with daily oral corticosteroids, with intravenous corticosteroids utilized in severe disease; however, recent data suggests that short-term use of intravenous cortcosteroids will allow a short-term improvement in strength, with no long-term change in outcome. More recent investigations suggest that early intervention with additional immunomodulatory agents will allow for a faster recovery, with less medication and disease sequelae. Use of methotrexate as an agent early in the disease course is becoming common place. Methotrexate, in conjunction with oral corticosteroids, allows a rapid improvement in symptoms, and allows for a more rapid reduction in corticosteroid dose. Methotrexate is considered as a steroid sparing agent, whether oral or intravenous corticosteroids are used. Additional immunomodulatory agents include the use of cyclosporine with or without methotrexate. Intravenous immunoglobulin has been reported to have benefit in resistant disease. There are exciting new agents which have great potential in treating JDM. Many of these agents are termed biologics and are being tested in adult myositis and juvenile arthritis. These include tumor necrosis factor (TNF)-alpha inhibitors, such as a chimeric monoclonal antibody to TNF-alpha, and a recombinant soluble human TNF receptor (p75)-Fc fusion protein. Many other new biological agents are also being tested in myositis.

2/3,AB/10 (Item 8 from file: 73)
DIALOG(R)File 73:EMBASE

11581900 EMBASE No: 2002153316
Editorial overview: Clinical therapeutics
Arend W.P.
Dr. W.P. Arend, Division of Rheumatology, Univ. Colorado Hlth. Sci. Ctr.
B115, 4200 East Ninth Avenue, Denver, CO 80262 United States
AUTHOR EMAIL: william.arend@uchsc.edu
Current Opinion in Rheumatology (CURR. OPIN. RHEUMATOL.) (United States
) 2002, 14/3 (201-203)
CODEN: CORHE ISSN: 1040-8711
DOCUMENT TYPE: Journal ; Editorial
LANGUAGE: ENGLISH

2/3,AB/11 (Item 9 from file: 73)
DIALOG(R)File 73:EMBASE

11241844 EMBASE No: 2001256103
A clinical and economic review of disease-modifying antirheumatic drugs
Gabriel S.E.; Coyle D.; Moreland L.W.
Dr. S.E. Gabriel, Mayo Foundation, 200 First St SW, Rochester, MN 55905
United States
AUTHOR EMAIL: gabriel.sherine@mayo.edu
PharmacoEconomics (PHARMACOECONOMICS) (New Zealand) 2001, 19/7
(715-728)
CODEN: PARME ISSN: 1170-7690
DOCUMENT TYPE: Journal ; Review
LANGUAGE: ENGLISH SUMMARY LANGUAGE: ENGLISH
NUMBER OF REFERENCES: 150

Rheumatoid arthritis is one of the most common chronic systemic
inflammatory diseases, affecting approximately 1% of the adult population.
Disease-modifying antirheumatic drugs (DMARDs) have been the mainstay of
treatment for rheumatoid arthritis when combined with physical therapy and
aspirin (acetylsalicylic acid) or nonsteroidal anti-inflammatory drugs.
Recently, a number of new biological therapies have been introduced for the
treatment of this condition and will have a major impact on the future
management of this disabling disease. In this review, we summarise data on
the efficacy and tolerability of the currently available DMARDs, including
gold compounds, antimalarials, penicillamine, cytotoxic drugs (azathioprine
and cyclophosphamide), sulfasalazine, methotrexate, leflunomide,
cyclosporin, anti-tumour necrosis factor agents, combination therapy and
apheresis. A literature review and quality assessment of economic
evaluations of DMARDs is presented, illustrating that there has been a
paucity of economic evaluations on these agents and showing the variable
quality of those studies that are available. The manuscript also addresses
the pharmacoeconomic implications of the new agents for rheumatoid
arthritis; the need for formal long term economic evaluations in order to
determine the cost effectiveness of these costly, but highly effective, new
treatments is emphasised.

2/3,AB/12 (Item 10 from file: 73)
DIALOG(R)File 73:EMBASE

11226302 EMBASE No: 2001240703
Arthritis therapy: A better time, a better day
Roth S.H.
S.H. Roth, Aging and Arthritis Program, Graduate Colleges, Arizona State
University, Tempe, AZ United States
Rheumatology (RHEUMATOLOGY (UK)) (United Kingdom) 2001, 40/6
(603-606)
CODEN: RUMAF ISSN: 1462-0324
DOCUMENT TYPE: Journal ; Editorial
LANGUAGE: ENGLISH
NUMBER OF REFERENCES: 30

2/3,AB/13 (Item 11 from file: 73)
DIALOG(R)File 73:EMBASE

07909244 EMBASE No: 1999382552
New therapeutic approaches for rheumatoid arthritis
Abdelaty E.M.; Schumacher H.R. Jr.
Dr. H.R. Schumacher Jr., Arthritis-Immunology Center, 151 K, VAMC,
University and Woodland Avenues, Philadelphia, PA 19104 United States
International Journal of Clinical Practice (INT. J. CLIN. PRACT.) (
United Kingdom) 1999, 53/7 (535-539)

CODEN: IJCPF ISSN: 1368-5031
DOCUMENT TYPE: Journal; Short Survey
LANGUAGE: ENGLISH SUMMARY LANGUAGE: ENGLISH
NUMBER OF REFERENCES: 26

Better understanding of the immunopathogenesis of rheumatoid arthritis
over the past few decades has promoted the innovation of new therapeutic
approaches targeting the disease more specifically. In addition,
refinements in the non-steroidal anti-inflammatory drugs (NSAID) may offer
advantages for patients with RA. This brief review reports and analyses
some of the important aspects of these new therapies available for
treatment of RA and in which situations you might consider each. Before
using any of these agents, physicians should become thoroughly familiar
with the package inserts.
?
? s extracorporeal (5n)(infliximab or etanercept)
42726 EXTRACORPOREAL
4228 INFLIXIMAB
2611 ETANERCEPT
S3 2 EXTRACORPOREAL (5N)(INFLIXIMAB OR ETANERCEPT)
? s s3 not s2
2 S3
13 S2
S4 2 S3 NOT S2
? t s4/3,ab/1,2

4/3,AB/1 (Item 1 from file: 155)
DIALOG(R)File 155:MEDLINE(R)
(c) format only 2004 The Dialog Corp. All rts. reserv.

12256872 PMID: 12603689
Steroid-refractory graft-vs.-host disease: past, present and future.
Carpenter Paul A; Sanders Jean E
Fred Hutchinson Cancer Research Center and University of Washington,
Department of Pediatrics, Seattle, WA 98109, USA.
Pediatric transplantation (Denmark) 2003, 7 Suppl 3 p19-31, ISSN
1397-3142 Journal Code: 9802574
Document type: Journal Article; Review; Review, Tutorial
Languages: ENGLISH
Main Citation Owner: NLM
Record type: Completed
Despite current standard preventive strategies that include optimizing
donor selection and the combination of methorexate and a calcineurine
inhibitor, acute and chronic GVHD remains a major barrier to successful
hematopoietic cell transplantation for a sizeable proportion of patients.
When acute and chronic GVHD become manifest a standard primary therapy
approach has been the addition of glucocorticoid therapy to a background of
calcineurine inhibition. When this approach fails patients with GVHD
require secondary therapy. Ideally, second-line agents should promote
transplantation tolerance so that the morbidity associated with prolonged
use of glucocorticoids and other immunosuppressive agents can be minimized.
Promising new agents or strategies which warrant further controlled
clinical trials include: mycophenolate mofetil, sirolimus, humanized or
chimeric monoclonal antibodies such as visilizumab, daclizumab and
infliximab, and *extracorporeal* photopheresis. Co-operative
studies are necessary to hasten the process of evaluating novel treatment
strategies for acute and chronic GVHD.

4/3,AB/2 (Item 1 from file: 73)
DIALOG(R)File 73:EMBASE
(c) 2004 Elsevier Science B.V. All rts. reserv.

12001984 EMBASE No: 2003113336
Steroid-refractory graft-vs.-host disease: Past, present and future
Carpenter P.A.; Sanders J.E.
P.A. Carpenter, Fred Hutchinson Cancer Res. Center, University of
Washington, Department of Pediatrics, Seattle, WA 98109 United States
Pediatric Transplantation (PEDIATR. TRANSPLANT.) (United Kingdom)
2003, 7/SUPPL. 3 (19-31)
CODEN: PETRF ISSN: 1397-3142
DOCUMENT TYPE: Journal ; Conference Paper
LANGUAGE: ENGLISH SUMMARY LANGUAGE: ENGLISH
NUMBER OF REFERENCES: 81

Despite current standard preventive strategies that include optimizing
donor selection and the combination of methorexate and a calcineurine
inhibitor, acute and chronic GVHD remains a major barrier to successful
hematopoietic cell transplantation for a sizeable proportion of patients.

When acute and chronic GVHD become manifest a standard primary therapy
approach has been the addition of glucocorticoid therapy to a background of
calcineurine inhibition. When this approach fails patients with GVHD
require secondary therapy. Ideally, second-line agents should promote
transplantation tolerance so that the morbidity associated with prolonged
use of glucocorticoids and other immunosuppressive agents can be minimized.
Promising new agents or strategies which warrant further controlled
clinical trials include: mycophenolate mofetil, sirolimus, humanized or
chimeric monoclonal antibodies such as visilizumab, daclizumab and
infliximab, and *extracorporeal* photopheresis. Co-operative
studies are necessary to hasten the process of evaluating novel treatment
strategies for acute and chronic GVHD.
? s extracorporeal/ti and (infliximab or etanercept)/ti
        15185 EXTRACORPOREAL/TI
         1206 INFLIXIMAB/TI
          542 ETANERCEPT/TI
    S5   0 EXTRACORPOREAL/TI AND (INFLIXIMAB OR ETANERCEPT)/TI
? s (infliximab or etanercept)/ti
         1206 INFLIXIMAB/TI
          542 ETANERCEPT/TI
    S6   1684 (INFLIXIMAB OR ETANERCEPT)/TI
? s s6 and pd>20001110
>>>One or more prefixes are unsupported
>>> or undefined in one or more files.
         1684 S6
      1632830 PD>20001110
    S7   751 S6 AND PD>20001110
? s s6 not s7
         1684 S6
          751 S7
    S8   933 S6 NOT S7
? s s8 and extracorporeal
          933 S8
        42726 EXTRACORPOREAL
    S9   0 S8 AND EXTRACORPOREAL
? s s8 and affinity adj chromatography
          933 S8
            0 AFFINITY ADJ CHROMATOGRAPHY
   S10   0 S8 AND AFFINITY ADJ CHROMATOGRAPHY
? s s8 and affinity
          933 S8
       368815 AFFINITY
   S11   4 S8 AND AFFINITY
? t s11/3,ab/all

 11/3,AB/1   (Item 1 from file: 155)
DIALOG(R)File 155:MEDLINE(R)

15841502 PMID: 14632813
 Infliximab for hidradenitis suppurativa.
Sullivan T P; Welsh E; Kerdel F A; Burdick A E; Kirsner R S
Department of Dermatology and Cutaneous Surgery, University of Miami
School of Medicine/Cedars Medical Center, 1400 NW 12th Ave. 6 South
Dermatology, Miami, FL 33136, USA.
British journal of dermatology (England) Nov 2003, 149 (5) p1046-9,
ISSN 0007-0963 Journal Code: 0004041
Document type: Journal Article
Languages: ENGLISH
Main Citation Owner: NLM
Record type: Completed
BACKGROUND: Hidradenitis suppurativa (HS) is a chronic disease
characterized by significant morbidity. Current medical therapies are only
minimally effective at treating the disease. Infliximab is a chimeric
monoclonal antibody with high *affinity* for tumour necrosis factor
(TNF)-alpha. TNF-alpha is known to induce proinflammatory cytokines and may
play an important role in the therapy of a number of disparate inflammatory
disorders. Infliximab has shown promise for the therapy of rheumatoid
arthritis and psoriasis. OBJECTIVES: Retrospectively to evaluate the
effectiveness of infliximab for the treatment of HS. METHODS: A
retrospective chart review was performed for patients who received
infliximab at the University of Miami Department of Dermatology. Patients
were contacted and asked retrospectively to rate their disease activity
immediately prior to and after therapy. RESULTS: Patients' self-reported
disease activity scores were significantly decreased (P = 0.0001, paired
t-test) following infliximab infusion. This correlated with
physician-observed clinical improvement. CONCLUSIONS: Infliximab is a
promising agent for the treatment of HS. These initial results suggest that
infliximab is associated with objective and subjective improvement in HS.
Further controlled studies of the efficacy of infliximab and its effect on

the course of the disease are warranted.

11/3,AB/2 (Item 2 from file: 155)
DIALOG(R)File 155:MEDLINE(R)

15008454 PMID: 12110154
How does *infliximab* work in rheumatoid arthritis?
Maini Ravinder N; Feldmann Marc
The Kennedy Institute of Rheumatology Division, Imperial College of Science Technology and Medicine, London, UK. r.maini@ic.ac.uk
Arthritis research (England) 2002, 4 Suppl 2 pS22-8, ISSN 1465-9905
Journal Code: 100913255
Document type: Journal Article
Languages: ENGLISH
Main Citation Owner: NLM
Record type: In Data Review
Since the initial characterization of tumor necrosis factor alpha (TNFalpha), it has become clear that TNFalpha has diverse biologic activity. The realization that TNFalpha plays a role in rheumatoid arthritis (RA) has led to the development of anti-TNF agents for the treatment of RA. Infliximab, a chimeric monoclonal antibody that specifically, and with high *affinity* , binds to TNFalpha and neutralizes the cytokine, is currently approved for the treatment of RA and Crohn's disease, another immune-inflammatory disorder. In addition to establishing the safety and efficacy of infliximab, clinical research has also provided insights into the complex cellular and cytokine-dependent pathways involved in the pathophysiology of RA, including evidence that supports TNFalpha involvement in cytokine regulation, cell recruitment, angiogenesis, and tissue destruction.

11/3,AB/3 (Item 3 from file: 155)
DIALOG(R)File 155:MEDLINE(R)

12055469 PMID: 12379627
Infliximab treatment for rheumatic disease: clinical and radiological efficacy.
St Clair E W
Department of Medicine, Division of Rheumatology, Duke University Medical Center, Durham, NC, USA. stcla003@mc.duke.edu
Annals of the rheumatic diseases (England) Nov 2002, 61 Suppl 2 pii67-9, ISSN 0003-4967 Journal Code: 0372355
Document type: Journal Article; Review; Review, Tutorial
Languages: ENGLISH
Main Citation Owner: NLM
Record type: Completed
Infliximab is a chimeric anti-tumour necrosis factor alpha (TNFalpha) monoclonal antibody with high *affinity* and binding specificity for human TNFalpha. Results from several well designed, controlled clinical trials show repeated infusions of infliximab with concomitant methotrexate (MTX) treatment can reduce the signs and symptoms of rheumatoid arthritis (RA). This combination of infliximab and MTX also slows the radiological progression of joint damage, decreases functional disability, and improves qualify of life. These remarkably positive results have led to the investigation of infliximab treatment for other rheumatic diseases. Recently, controlled studies have shown treatment with infliximab can benefit patients with active spondyloarthropathy. TNFalpha has fast become an important therapeutic target in rheumatology.

11/3,AB/4 (Item 4 from file: 155)
DIALOG(R)File 155:MEDLINE(R)

11367282 PMID: 11458980
Immunomodulatory effects of *etanercept* (TNFR:Fc) and its use in a patient with Crohn's disease.
Srivastava M D
Department of Pediatrics, MetroHealth Medical Center, Cleveland, Ohio 44109, USA.
Research communications in molecular pathology and pharmacology (United States) Jul 2001, 109 (1-2) p125-41, ISSN 1078-0297 Journal Code: 9437512
Document type: Case Reports; Clinical Trial; Journal Article
Languages: ENGLISH
Main Citation Owner: NLM
Record type: Completed

Designer drug etanercept (TNFR:Fc) is an inhibitor of TNF-alpha that
binds with greater *affinity* than membrane receptors. Its full
immunomodulatory effects are unknown. Approved for rheumatoid arthritis,
its therapeutic potential in Crohn's disease has yet to be explored. We
describe the course of a steroid-dependent patient with Crohn's disease
given etanercept, and its effects on cytokine protein and mRNA expression
and transcription factor activity in human leukocytes. Etanercept 25 mg
s.c., was given twice weekly for 1 month. Weekly ESR, disease activity
index, prednisone requirement, and serum cytokines were determined. In
vitro, effects of physiologic concentrations of etanercept on cytokine
protein and mRNA, and NFKB and GR transcription factor activity, were
determined using MOT and U937 cell lines and peripheral blood mononuclear
cells. Rapid clinical, biochemical, and immunologic improvement occurred,
but obstruction due to stricture developed after 4 weeks. In vitro,
constitutive and stimulated production of TNF-beta, IL-1beta, MIP-1beta,
and IL-8 by normal mononuclear cells declined with etanercept, detectable
TNF-alpha increased. MOT TNF-alpha expression tripled, mRNA for IL-12 p40
doubled, GR activity declined in U937 cells, NFKB was unaffected.
Etanercept has complex immunomodulatory effects, and may be useful in
Crohn's disease, but acutely decreased inflammation could worsen stricture.
? ds

Set Items Description
S1 14 (ETANERCEPT OR INFLIXIMAB) AND APHERESIS
S2 13 RD (unique items)
S3 2 EXTRACORPOREAL (5N)(INFLIXIMAB OR ETANERCEPT)
S4 2 S3 NOT S2
S5 0 EXTRACORPOREAL/TI AND (INFLIXIMAB OR ETANERCEPT)/TI
S6 1684 (INFLIXIMAB OR ETANERCEPT)/TI
S7 751 S6 AND PD>20001110
S8 933 S6 NOT S7
S9 0 S8 AND EXTRACORPOREAL
S10 0 S8 AND AFFINITY ADJ CHROMATOGRAPHY
S11 4 S8 AND AFFINITY
? s s6 and immobilized/ti
1684 S6
11263 IMMOBILIZED/TI
S12 0 S6 AND IMMOBILIZED/TI
? logoff
01jul04 11:58:54 User217743 Session D640.5
$0.94 0.293 DialUnits File155
$1.47 7 Type(s) in Format 4 (UDF)
$1.47 7 Types
$2.41 Estimated cost File155
$11.39 1.162 DialUnits File73
$13.50 5 Type(s) in Format 3 (UDF)
$18.90 7 Type(s) in Format 4 (UDF)
$32.40 12 Types
$43.79 Estimated cost File73
OneSearch, 2 files, 1.456 DialUnits FileOS
$1.25 TELNET
$47.45 Estimated cost this search
$57.73 Estimated total session cost 3.369 DialUnits
Logoff: level 04.11.00 D 11:58:54